

COMMENTARY

Prognostic indicators in patients presenting with acute cardiogenic pulmonary edema treated with CPAP: it's not the acid that matters, it's back to basics

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See related research by Aliberti *et al.*, <http://ccforum.com/content/14/6/R196>

Abstract

Several prognostic markers have been identified for patients admitted with acute cardiogenic pulmonary edema. Most of the markers are based on clinical risk scores. Unlike hypercapnic respiratory failure, acidosis is not an adverse predictor in these patients. Hemodynamic variables that assess pathophysiological mechanisms may be more helpful to guide appropriate management.

In this issue of *Critical Care*, Aliberti and colleagues [1] examine the question of whether acidemia predicts outcome in patients admitted with acute cardiogenic pulmonary edema (ACPE) and treated with continuous positive airway pressure (CPAP). The authors performed a retrospective observational study in a large cohort of patients admitted to the emergency department with ACPE and treated with CPAP. The authors compared treatment failure, defined as a switch to bi-level or invasive ventilation or in-hospital mortality, in two matched groups of patients with and without acidemia on admission. The main conclusion was that neither admission acidemia nor type was an adverse prognostic marker.

Acute heart failure syndromes (AHFSs) are the most common reason for hospital admission in patients older than 65 years, over 50% of whom present with ACPE [2], and up to 10% of patients with ACPE will not survive

their admission [3]. A number of prognostic factors have been identified: age, systolic blood pressure (SBP), brain natriuretic peptide or troponin rise, hyponatremia, renal dysfunction, previous ischemic heart disease, ejection fraction, and function at discharge. However, most of these variables have been used across the whole spectrum of AHFSs and relate to longer-term outcome [4,5]. Focusing on ACPE only, the 3CPO Trialists identified patients at immediate risk of death and in need of intervention on presentation; a simple score based on age, SBP, and the ability to obey commands predicted early mortality [6].

In acute exacerbations of chronic obstructive pulmonary disease, acidosis is a strong predictor of adverse outcome [7]. Why does it not have similar prognostic implications in ACPE? Acidosis in hypercapnic respiratory failure is a direct reflection of organ dysfunction, in particular the failure of the lungs to ventilate the alveoli adequately. In contrast, in ACPE, acidosis is only an indirect consequence of cardiac dysfunction caused by a combination of factors, including edema-impaired gas exchange and tissue hypoperfusion. The absence of acidosis is unusual in patients with ACPE [1]. It is therefore not surprising that acidosis is a poor discriminator of outcome. More direct indicators of cardiac function, such as cardiac power output (CPO), which is derived from the product of cardiac output and mean arterial blood pressure, have been shown to be powerful prognostic indicators in patients with AHFS, chronic heart failure (CHF), or cardiogenic shock [8-10].

Clinical risk scores, a product of statistical modeling, are a popular tool to identify prognostic markers. However, most do not pass the acid test of clinical utility, and this is evidence that a change in risk score correlates with changes in patient outcome. Furthermore, most risk scores do not provide any information on underlying pathophysiology.

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Differentiating the pathophysiological processes underpinning the different AHFSs to provide appropriate and rational treatment is an alternative approach to risk-stratify patients with ACPE. Cotter and colleagues [11] suggested four major clinical AHFSs: (a) pulmonary edema, (b) cardiogenic shock, (c) hypertensive (HTN) crisis, and (d) exacerbated systolic CHF. By plotting CPO as a marker of cardiac contractility against systemic vascular resistance (SVR), the authors demonstrated that 95% of patients could be categorized accurately into one of these syndromes [12]. Understanding the pathophysiological mechanisms is key to management; for example, patients with cardiogenic shock and those with pulmonary edema demonstrate a similar presentation with pulmonary congestion, clammy extremities, low cardiac output, and high wedge pressure. However, the pathophysiologies are very different and the treatments are almost opposite. The former is characterized by low CPO and low SVR that requires inotropic support and occasionally peripheral vasoconstrictors, and the latter is characterized by extreme neurohormonal activation with higher values of CPO and very high SVR, necessitating aggressive peripheral vasodilation. According to this model, exacerbated systolic CHF, HTN crisis, and ACPE may be viewed as a continuum of progressive neurohormonal activation leading to an increase in SVR with progressive recruitment of cardiac power reserve [8]. The objective of treatment is then to optimize the SVR and thus the cardiac performance can be moved toward more optimal loading conditions to effect greater power output [13]. The need for invasive hemodynamic monitoring is a limitation, but the advent of non-invasive tools could improve diagnosis, risk stratification, and management of patients with AHFS.

The etiology of heart failure and the cardiac rhythm influence the acute cardiac response to CPAP. In patients with dilated cardiomyopathy, CPAPs lead to a reduction in cardiac volumes, whereas in ischemic cardiomyopathy, there were small increases [14]. Similarly, atrial fibrillation resulted in a fall in cardiac index but there was a small rise in sinus rhythm [15].

CPAP, with its unique combination of respiratory and hemodynamic effects, will remain a cornerstone of the symptomatic treatment of ACPE. Future management should look beyond clinical risk scores to an understanding of etiology and pathophysiology in each patient to provide optimal treatment for this deadly condition.

Abbreviations

ACPE, acute cardiogenic pulmonary edema; AHFS, acute heart failure syndrome; CHF, chronic heart failure; CPAP, continuous positive airway pressure; CPO, cardiac power output; HTN, hypertensive; SBP, systolic blood pressure; SVR, systemic vascular resistance.

Competing interests

The authors declare that they have no competing interests.

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